Balance disorders/PGS

Introduction

Balance means the ability to maintain the position of the body and its parts in space. It depends on constant afferentation from the somatosensory, vestibular and visual systems, in the processing of this information in the brainstem, cerebellum, basal ganglia and some parts of the cortex. Dysfunction of any of these areas of the nervous system leads to a relatively specific balance disorder that can be distinguished by a careful history and clinical examination.

The history of balance disorders must include the following information:

- type of balance disorder, ameliorating and aggravating factors
- moments of provocation and the beginning of a feeling of instability (acute, gradual)
- duration of progression (minutes, days, months, years)
- course (continuous, progressive, episodic), in the case of episodic, the duration of the attack
- accompanying symptoms (vegetative, auditory, other neurological)
- history of internal and psychiatric comorbidities and used medication

Already from the anamnesis and clinical examination, it is possible to distinguish:

1. vertigo, which indicates a lesion of the vestibular system
2. ataxia caused by lesions of the cerebellum, proprioceptive pathways or the vestibular system
3. stability disorders caused by lesions of the basal ganglia, frontal cortex or their connections
4. non-specific or pre-syncopal states of a feeling of imbalance, which are usually caused by cardiovascular, metabolic or vegetative causes.

Distinguishing the causes of balance disorders

Vertigo

Vertigo is the illusion of movement of the body or environment, which can be linear or rotational. Patients perceive it as a feeling of a chain carousel, sensations of swinging or pulling to the side. Vertigo is most often caused by dysfunction of the peripheral (labyrinth of the inner ear, **vestibular nerve**(or central (vestibular nuclei of the brainstem and their connections) vestibular system. Less often, it can be caused by a lesion of the vestibular nuclei of the cerebellum, it rarely has a cortical-epileptic origin, mostly in the frontal or parietotemporal region. It is often accompanied by vegetative symptoms: **nausea**, **vomiting**, and pallor. It is a subjectively very unpleasant experience.

Vertigo must be distinguished from non-vestibular dizziness and other causes of balance disorders, and it is also necessary to distinguish between the vertigo of peripheral and central origin. It should be noted that vertigo of peripheral origin is caused by asymmetry of the function of the labyrinths or vestibular nerves (i.e. unilateral hyperfunction or hypofunction). When both labyrinths are symmetrically affected (the term bilateral vestibulopathy is used), vestibular ataxia and oscillopsia (decreased visual acuity when moving the head) occur.

Vertigo supports:
▪ spontaneous sensations of traction or rotation accompanied by nausea and vomiting ▪ symptoms are induced by changing the position of the head ▪ blurred vision during rapid head movements (e.g. when walking) ▪ spontaneous, visual or positional nystagmus ▪ tonic deviations of the limbs ▪ **vestibular ataxia** ▪ associated trunk or auditory symptoms

Nystagmus

Nystagmus (from the Greek nystagmein - drooping of the head when falling asleep sitting up) is an oscillatory movement of the eyeballs, usually biphasic with a slow and fast component. The slow component is the basic symptom of the so-called static vestibular imbalance, which is caused by the asymmetry of the function of the vestibular apparatus, pushing the bulbs to the side of the weaker labyrinth. The fast component is a refixation saccade that returns the eye to its starting position. The fast phase is a reflex action generated by the **reticular formation** brainstem (paramedian pontine reticular formation – PPRF – for horizontal saccades and n. rostralis interstitialis fasciculi longitudinalis medialis – riMLF – for vertical and rotational saccades). Rhythmic movement is created by constant repetition of both components of nystagmus.

We arbitrarily determine the direction of nystagmus according to the fast component. During the examination, we describe the plane in which the nystagmus beats (oscillates) - it can be horizontal, vertical or rotational. Peripheral vertigo is characterized by rhythmic, horizontal-rotational nystagmus, in which we distinguish three quantitative degrees, reflecting the degree of asymmetry of the function of both vestibular apparatuses: I. degree appears only when looking in the direction of the fast component, II. degree even when looking straight on (primary position of the bulbs) and III. an extra degree even when looking in the direction of the slow component (looking against the direction of the nystagmus). *This dependence of intensity on the direction of view, the so-called Alexander's law*, is typical for peripheral vertigo of labyrinthine origin. This type of nystagmus is also significantly dampened by visual fixation, so it can be accentuated by closing the eyes (we examine by palpation through the eyelids) or by wearing Frenzel glasses, which make fixation impossible (they have magnifying glasses and internal lighting that dazzles the patient).

Nystagmus in a vertigo of central origin is variable, tends to be dysrhythmic (alternates larger and smaller amplitude), often has a vertical component, and the direction can change even during the examination. The most common type of central nystagmus is the so-called regularly changing visual nystagmus. It is first-degree nystagmus, always beating in the direction of gaze (when looking to the right, the nystagmus is right-sided, when looking to the left, it is left-sided, so the direction is reversed), in the primary position of the bulbs, nystagmus is not present. This nystagmus is practically always present even with lighter degrees of intoxication, but it belongs to the signs of intoxication with centrally acting drugs in general. It is a valuable objective symptom of an overdose of antiepileptics, neuroleptics, and antidepressants. The pathophysiological basis is the dysfunction of the Purkinje cells of the flocculus, which cannot maintain the eccentric position of the bulb when viewed laterally.

Vertical downward beating nystagmus represents the so-called **downbeat nystagmus syndrome**, topically specific for lower **trunk** lesions. It is always necessary to rule out **Arnold-Chiari malformation** first, but it is also accompanied by intoxication and vascular lesions.

Another type of central nystagmus is dissociated nystagmus, in which each eye oscillates differently. The most common example of this type of nystagmus is the so-called internuclear ophthalmoplegia – when looking to the side, nystagmus appears on the abducting eye, the adducting eye does not close and the nystagmus is not visible. The syndrome can be unilateral or bilateral. It typically occurs in patients with **multiple sclerosis of the cerebrospinal** cord when fibers of the fasciculus longitudinalis medialis are affected at the level of the pontine. Rarer is dissociated nystagmus, which has coarse, slow and irregular oscillations when viewed on one side, and fine, fast and regular oscillations when viewed on the other side - the so-called Bruns-Stewart dissociation. It sometimes occurs with cerebellar lesions, typically with cerebellar abscesses of otogenic origin as a complication of mastoiditis.

Tonic deviations

They are also a reflection of static vestibular imbalance. In vertigo of peripheral origin, their direction is identical to the slow component of nystagmus (they are directed towards the weaker vestibular apparatus), therefore the peripheral vestibular syndrome is sometimes called harmonic. *Hautant's test* is performed sitting with the back supported, eyes closed and the upper limbs on the forearms. The deviation of the limbs to the side in 30 seconds is monitored. Deviation of standing from the vertical has a similar cause. It is true that the patient tends to fall behind the weaker labyrinth (ear), so the direction of the fall changes when the head is turned. There is also a deviation from a straight direction when walking. A more sensitive test is *the Unterberger test* when the patient is allowed to march in place with forearms and eyes closed for at least 30 seconds. With vestibular asymmetry, we observe a deviation of more than 45°. Central vestibular syndrome causes tonic deviations and falls without lateral predilection, and is therefore sometimes called disharmonic.

Vestibular ataxia

Vestibular ataxia is a disorder of balance and coordination of movements, which is not observed lying down, but only when standing and walking, especially when the eyes are closed.

Deterioration of balance with closed eyes is called Romberg's sign, which is observed in vestibular and mainly in sensory ataxia.

Conversely, it is negative for cerebellar ataxia.

Associated auditory or trunk symptoms

We examine the hearing as an orientation by rubbing the fingers gradually over both ears of the patient. Hearing impairment associated with vertigo suggests its peripheral origin. From the trunk symptoms, we will examine oculomotor skills, the function of other cranial nerves, the cerebellum and long motor and sensory pathways. The finding of trunk symptoms along with vertigo is indicative of its central origin.

Special exams

In milder unilateral vestibular lesions that do not cause spontaneous nystagmus, we examine the so-called *dynamic vestibular function* using the head-impulse test, which consists of passive head movements in the planes of the semicircular canals while visual fixation of one point. Impairment of the vestibulo-ocular reflex manifests itself in the insufficient compensatory movement of the bulbs against head movement when moving the head towards the affected labyrinth, which must be compensated by a compensatory saccade.

A very sensitive test for a unilateral vestibular disorder is the *caloric test* – injection of warm and cold water into the ear canal, which induces nystagmus, the intensity of which is recorded on both sides and subsequently compared. If positional vertigo is suspected, we perform *positional tests* – Dix-Hallpike manoeuvre – during which we turn the patient's head to a 45-degree angle and quickly place him in this way with his head over the bed, we monitor the development of vertigo and nystagmus, which typically comes with a latency of several seconds and up to a minute fade away. We also perform the test with the head facing forward and turned 45 degrees to the other side.

Auxiliary examinations

Audiometry

It objectives hearing impairment, the presence of which indicates a peripheral disability.

Stem evoked potentials

They objectify the impairment of the vestibular pathway.

CT scan of the brain

We indicate central and post-traumatic vestibular syndrome. It will help rule out serious lesions of the inner ear, and lesions of the cerebellum (malacia, hemorrhage, atrophy), and can show older trunk malacia, tumor of the pontine angle and diffuse ischemic lesions.

MRI of the brain

It is more accurate in the diagnosis of multiple sclerosis, trunk malacia and tumors of the pons.

Liquor

It is necessary to examine if an inflammatory cause or multiple sclerosis is suspected.

X-ray of the cervical spine

It will show degenerative changes when cervicogenic imbalance is suspected. However, the examination has little informative value, as the finding of degenerative changes does not mean confirmation of a causal connection with a balance disorder.

Sonography of cerebral arteries

demonstrates hemodynamic disorders or congenital abnormalities of the brainstem.

Tab. 1: Distinguishing between peripheral and central vestibular syndrome

	Peripheral	**Central**
Intensity, character	Strong, proportional to nystagmus, intermittent	Mild to moderate, continuous
Nystagmus	Unidirectional, horizontal, gaze fixation inhibits it	Verticing, changing direction, dissociated, gaze fixation without effect
Tonic deviations	One way – harmonious	In different directions – disharmonius
Compensation	Relatively fast- habituation and nystagmus fatigue	Difficult, often chronic
other symptoms	Auditory (tinnitus, hypacuse)	Tribal (dysarthria, diplopia)

Ataxia

Ataxia is dyscoordination or clumsiness of movements that is not the result of muscle weakness. This is a failure of the plan, measurement and monitoring of the trajectory of movements. It can affect eye movements, speech, limbs, trunk, standing and walking and is caused by lesions of the vestibular apparatus, cerebellum or proprioceptive pathways.

Clinical examination

Taxi is examined on the upper limbs with the finger-nose test, when we let the patient alternately touch his nose with his index finger and our finger, whose position we change, then we examine the finger-nose test with closed eyes. On the lower extremities, we examine the taxi with the heel-knee test, where the patient touches the heel of the knee of the other lower extremity without visual control and then slowly moves the heel down the lower leg. Trunk ataxia is investigated by standing stability test, first with slightly straddled (stance I), then supine (stance II) and finally standing supine with closed eyes (stance III). We evaluate deviations to the sides (titubation), possibly if there is a tendency to fall in a certain direction.

Cerebellar ataxia is supported by:
▪ clumsiness of upper and lower limbs

- patient reports feeling "little drunk"
- it doesn't get much worse after closing the eyes
- dysarthria character of saccadic speech
- during the toe-nose or heel-knee test, there is an intentional tremor (always just before the target) and hypermetria (missing the target)
- passivity (increased excursion of the upper limbs when walking or turning the trunk by the examiner)
- dysdiadochokinesis (inability to perform rapid alternating limb movements such as turning the arms into pronation/supination)
- may be accompanied by nystagmus
- asynergy of movements (they are jerky, divided into a sequence of partial movements, not continuous)

Sensory ataxia is supported by:

- history of impaired stability in dim light (it also worsens when eyes are closed)
- paresthesia and numbness of lower limbs
- worse clinical findings on the lower than on the upper limbs
- reduced posture, mobility and paresthesia on the lower limbs (examined with a tuning fork)
- hyporeflexia

Auxiliary investigative methods in cerebellar syndrome

CT scan of the brain

which can show atrophy or structural lesions of the cerebellum (tumor, malacia, hemorrhage, atrophy).

MRI of the brain

which is more accurate in depicting cerebellitis and multiple sclerosis (MS).

Copper metabolism screening

an ophthalmological examination focused on the detection of the Keyser-Fleyscher ring must be performed in patients younger than 45 years to rule out Wilson's disease.

Genetic testing

indicated for suspected spinocerebellar ataxia, Friedreich ataxia or Wilson's disease.

Paraneoplastic antibodies

(anti-Yo) when paraneoplastic involvement is suspected.

Examination of the cerebrospinal fluid

to rule out an inflammatory cause and MS and *vitamin E levels*.

Auxiliary examination methods in sensory ataxia

The causes of sensory ataxia are numerous and can be located in the peripheral nerves (neuropathy) or in the long pathways of the spinal cord, especially the posterior cords that carry proprioceptive information (myelopathy). The following will help in the diagnosis: *EMG* to confirm axonal or demyelinating lesions of the peripheral nervous system.

Liquor

to rule out chronic immune demyelinating polyneuropathy and infectious causes.

Paraneoplastic antibodies

(anti-Hu) when paraneoplastic involvement is suspected.

MRI of the spinal cord

it shows atrophy or structural involvement of the posterior cords (compression by tumorous masses, multiple sclerosis, vascular malformation, spinal cord ischemia, vasculitis).

Further, the search for the cause of polyneuropathy (vitamin B12 level, blood glucose, TSH) will help.

Impairment of stability in lesions of the frontal pathways, basal ganglia and their connections

it is primarily caused by the loss of postural reflexes and impaired initiation of movements - especially walking. We examine postural reflexes with a pull test, where we assess the patient's ability to compensate for deviation from balance by pushing backwards. The physiological response is to bend the knees. We also carefully examine walking and the presence of other symptoms of Parkinson's syndrome (akinesia, rigidity, tremors). This type of stability disorder is discussed in more detail in the chapter - Parkinson's syndrome.

Imaging methods (CT, MRI of the brain), which can distinguish structural abnormalities in the area of the basal ganglia, multi-infarct involvement of the white matter (Binswanger's disease) and normotensive hydrocephalus, are particularly useful as auxiliary examinations.

Frontal stability and gait disorder are supported by:
▪ widened base walking along with fear of falling and short steps ▪ improvement of stability even with minimal support (holding the examiner's finger) ▪ hesitation (hesitation) when starting to walk ▪ freezing and deterioration of stability in turns while walking ▪ walking in anteflexion, with reduced synkinesis ▪ finding other symptoms of Parkinson's syndrome ▪ finding symptoms indicative of diffuse white matter lesions (pyramidal irritation symptoms, axial syndrome, pseudobulbar syndrome, grasping reflex) ▪ in the pull-test deviation, the center of gravity will not be balanced by bending the knees but will lead to a pulse (jumping with short steps) and/or to a fall

Presyncopal states and non-specific feelings of imbalance

(emptiness in the head, uncertainty, feeling as if on water) do not have a vestibular character and can be caused by an insufficient supply of oxygen and nutrients to the brain in cardiovascular, hematological or metabolic disorders, or by abnormal processing of sensory inputs or abnormal experiencing internal states in psychiatric disorders. It is always necessary to remember that disorders of the cardiovascular system can also cause central vestibular syndrome, especially in older patients in the field of atherosclerotic changes in the vertebrobasilar and river bed and branches of the circle of Willis.

If a presyncopal state is suspected, it is necessary to perform an *ECG*, examination, measure *blood pressure* while lying down and after standing, and perform a *biochemical examination* (glycemia, mineralogram, renal parameters, TSH, inflammatory markers) and a *blood count*.

Sonography of cerebral arteries can demonstrate bilateral hemodynamically significant carotid stenosis or diffuse atherosclerotic changes in the vertebrobasilar basin.

The above examinations must also be performed in case of the central vestibular syndrome!

We will perform an EMG if the tetanic syndrome is suspected, as well as an examination level of *Mg and Ca*.

Instability not caused by a lesion of nerve structure is evidence of:
▪ the patient is unable to accurately describe his instability ▪ it is often a strange, hard-to-specify sensation in the head ▪ history of diabetes, cardiovascular disorders or thyreopathy ▪ positive psychiatric history ▪ newly started BP-lowering drugs ▪ a finding of increased neuromuscular excitability (Chowstk's phenomenon, lively reflexes)

Clinical units

Disorders of the peripheral vestibular system (both acute and chronic)

If the peripheral vestibular syndrome is suspected, consultation with an ENT specialist is necessary.

Benign paroxysmal positional vertigo

It is a very common cause of the peripheral vestibular syndrome. There is a history of repeated attacks of vertigo of the peripheral type, lasting less than 1 minute, induced by a change in the position of the head. They typically appear with a latency of several seconds after lying down and after verticalization. Patients also complain of slight instability when walking. The cause is the release of calcium carbonate crystals from the otolith system and their travel into the semicircular canals (most often into the back), which leads to irritation of the hair cells when moving

the head. It occurs most often in the 6th decade, more in women, the risk factor is head trauma and previous viral labyrinthitis. We establish the diagnosis using the Dix-Hallpike position test. The therapy of choice is positioning manoeuvres (Semont's or Epley's), during which the crystals move from the semicircular canals to the utricle, where they do not cause problems.

Epley manoeuvre (https://www.youtube.com/watch?v=ZqokxZRbJfw) - YouTube presentation (English)

Acute inflammatory vestibulopathy

In the case of viral **vestibular neuronitis** (neurolabyrintitis) vertigo develops within a few hours, reaches its maximum in about 24 hours and then gradually subsides, but complete adjustment only occurs within 1–3 months. In about 50% of cases, it is preceded by a viral disease, sometimes it occurs epidemically in families. It is not accompanied by significant hearing impairment. This benign entity must be differentiated from **zoster oticus**, which is caused by the varicella-zoster virus. It usually starts with an earache, followed by the seeding of a vesicular eruption in the area of the external auditory canal and on the eardrum (sometimes it can only be seen otoscopically - in case of suspicion, an ENT examination is necessary). At any time during the course, damage to the VII and VIII cranial nerves can occur, which can be irreversible. Also **borreliosis** in stage II. can cause disorders of the cranial nerves, typically a bilateral lesion of VII. nerve, less often lesions of the oculomotor or vestibulocochlear nerve. The diagnosis of herpes zoster and borreliosis is confirmed by a positive cerebrospinal fluid examination (cytoprotein association with mononuclear pleocytosis, detection of the pathogen microscopically or by PCR, detection of specific antibodies). **Bacterial labyrinthitis** is rare today and can be a complication of otitis media or osteomastoiditis. It usually has a rapidly developing severe course. In anamnesis, it is possible to find pain in the area of the ear or mastoid process. The diagnosis is confirmed by an otoscopic examination and/or a CT pyramid.

Meniere's disease

Etiology and clinical course

It is caused by an increase in pressure in the endolymphatic system, which leads to repeated ruptures of the membrane separating the peri- and endolymphatic space. It is manifested by repeated attacks of vertigo, a feeling of fullness in the affected ear, fluctuating hearing impairment and tinnitus, which typically lasts several minutes to hours. The disease occurs more often in women, between the ages of 30 and 50. a year. Attacks occur at irregular intervals of a week to several years, the hearing impairment is usually reversible in the early stages, later remains a residue in the form of hearing impairment in the low-frequency area demonstrable by audiometry. Over 20 years of disease duration, up to 20% of patients may have bilateral impairment of hearing and vestibular function.

Meniere 's syndrome is a set of symptoms similar to *Meniere's disease*, but with a known etiology affecting the permeability of blood vessels. The most common are degenerative diseases of the cervical spine, endocrinological diseases, allergic reactions, and infections.

Clinical course

A triad is typical: **tinnitus, rotational vertigo,** and **hearing loss**. Symptoms are typically **unilateral**. Accompanying symptoms such as nausea, vomitus, and balance disorders may appear. Attacks last minutes, hours, and occasionally even days. They fade away slowly. Repeated seizures can lead to hearing loss.

Diagnostics

We establish the diagnosis based on:

- history of at least two typical attacks (dg. cannot be reliably determined after the first attack),
- proof of a shift in the hearing threshold by 10 dB in 2 different frequencies during at least one examination,
- by ruling out another cause.

diff. dg.

- vestibular schwannoma
- Multiple sclerosis
- circulatory disorders of the CNS
- CVS disease

Therapy

Therapy consists of dietary measures – reducing salt in the diet, limiting smoking and caffeine, as well as pharmacotherapy with diuretics or betahistine. In the late stages, local endolymphatic application of gentamicin is considered. Vestibular rehabilitation is appropriate.

Post-traumatic Vertigo

Dizziness after head trauma is very common and can occur as a result of damage to the peripheral vestibular apparatus, contusion of the trunk or injury to the cervical spine. Post-traumatic vertigo must be examined with a brain CT scan, which will show a possible *fracture of the pyramid* with possible consequences of tears in the membranous labyrinth or laceration of the vestibular nerve. We always think of a base fracture when hemorrhagic or cerebrospinal fluid otorrhea is found. *Labyrinth contusion* often follows sharp blunt blows to the ear region. In the pathophysiology, microhemorrhages and damage to hair cells, which are not visible on the CT brain, are most likely to be used. Contusions of the labyrinth are often associated with perforation of the tympanic membrane. *Perilymphatic fistula* it can occur with fractures of the base, but also with blunt trauma with a sudden change in the pressure of the cerebrospinal fluid and subsequent leakage of perilymph from the inner ear, as well as with barotrauma, strong noise or increased abdominal pressure. The development is sudden and is usually accompanied by a loud popping sound in the ear. The most common vertigo following an injury is *benign positional vertigo*, which occurs with a certain latency. It is important to distinguish non-vestibular dizziness within the post-concussion *syndrome*, which includes dizziness, headache, irritability, impaired concentration and forgetfulness, and also within the *whiplash injury of the cervical spine* caused by sudden sharp flexion and extension of the spine, most often in car accidents, which causes pain in the cervical spine and head. It is also important to differentiate central vertigo at *trunk contusion* from ischemic or hemorrhagic changes. The diagnostic method of choice in this case is MRI.

Tumors of the pontine angle

They are mostly benign and grow very slowly. In 80% of cases, it is a vestibular schwannoma *(previously a statoacoustic neuroma)*, in the remaining cases, it is most often a meningioma or *cholesteatoma*. A neurinoma is usually first manifested by unilateral hearing loss or tinnitus, gradually adding a balance disorder, which tends to have a non-specific character. In an advanced stage, other cranial nerves passing through the pontine angle (trigeminus, n. facialis) are affected. In diagnosis, MRI takes precedence. The therapy is surgical, preferably open surgery, an alternative is stereotactic gamma knife operation. Since schwannoma it grows very slowly, we choose a conservative procedure - observation - for older patients and smaller tumors.

Otosclerosis

It is caused by the immobility of the stirrup, which begins to manifest itself mostly in the 2nd decade and often has a hereditary component. It manifests as hearing impairment with recurrent vertigo of the peripheral type. In the differential diagnosis, Meniere's disease must be distinguished (positive family history, onset at an earlier age, conductive hearing disorder).

Toxic vestibulopathy

Alcohol in acute intoxication can cause cerebellar and vestibular ataxia with vertigo and nystagmus. However, when nystagmus is found after an injury in an intoxicated person, a more serious cause of this condition must first be ruled out. **Aminoglycosides, salicylates, quinine, quinidine** and **cis-platinum** are also vestibulotoxic.

Central vestibular and cerebellar disorders with mainly acute development

Drug intoxication

Cerebellar syndrome, sometimes accompanied by confusion, can be caused by alcohol intoxication, sedative-hypnotics such as **benzodiazepines** or **barbiturates** or anticonvulsants - typically **phenytoin** or **carbamazepine**. Intoxication can also occur by mistake, by mistaking a weaker for a stronger dose of the drug (e.g. Rivotril 0.5 mg for a 2 mg package). The levels of these substances can be determined in plasma and/or urine, which is important in confirming the suspicion of intoxication.

Wernicke's encephalopathy

An acute disorder resulting from a deficiency of vitamin B1 (thiamine), which can occur with chronic malnutrition of any origin (most often in alcoholics, also during pregnancy, for example). An acutely formed triad manifests itself: ataxia, ophthalmoplegia and a delirious state. Oculomotor problems can be of the nature of bilateral abducent paresis or gaze paresis, pupillary abnormalities and nystagmus also occur. The diagnostic test is the administration of 100 mg of thiamine intramuscularly, after which the oculomotor skills will be adjusted within a few hours. Ataxia resolves with a latency of weeks to months, often with a residual.

Trunk ischemia

Vertebrobasilar insufficiency

(VBI) is one of the most common causes of vestibular syndrome in elderly patients. It is caused by brainstem ischemia, begins suddenly, often following dehydration or BP decompensation. Central vestibular syndrome is often accompanied by other trunk symptoms (dysarthria, diplopia, limb ataxia), but vertigo can be an isolated symptom. VBI is a transient ischemic attack in the vertebrobasilar basin, the symptoms disappear within 24 hours (typically within 1-2 hours), but they may recur in a short time. VBI is therefore a warning of an impending stroke. The etiology is most often thrombotic or embolization stenosis of the arteries of the vertebrobasilar basin (a. vertebralis, a. basilaris, a. labyrinthi, a. cerebelli anterior inferior – AICA, or posterior inferior – PICA). Less frequent

causes are compression of the vertebral artery in degenerative changes of the cervical spine, **subclavian steal syndrome** in stenosis of the subclavian artery (when the flow in the vertebral artery is reversed) or hemodynamic disorders.

Stroke

Symptoms lasting more than 24 hours will appear in the VB of the basin. In the case of a labyrinthine infarction, a sudden peripheral vestibular syndrome occurs, which is long-lasting and causes hearing impairment, which is irreversible. Even with VBI, ischemia of the labyrinth artery can sometimes dominate and manifest itself as a peripheral vestibular syndrome. PICA ischemia causes **Wallenberg's syndrome** (most often caused by proximal occlusion of the vertebral artery): central vestibular syndrome, diplopia, homolateral facial sensory impairment, Horner's syndrome, paresis of the soft palate, limb ataxia, and contralaterally dissociated heat and pain sensory impairment. In diagnosis, the method of choice is MRI, which better visualizes the posterior fossa area.

Bleeding in the posterior fossa

They are most often caused by arterial hypertension, less often the cause is arteriovenous malformation, tumor, trauma or coagulopathy. It is manifested by a sudden, severe headache, accompanied by nausea, vomiting, vertigo and ataxia, and sometimes by a disturbance of consciousness. A life-threatening complication is often compression of the trunk with the development of spastic paresis of the lower limbs and acute hydrocephalus. CT is the diagnostic method of choice and surgical evacuation of the hematoma is a life-saving procedure.

Infectious diseases

The acute cerebellar syndrome can rarely be caused by an infectious cause - viral encephalitis (varicella, mumps, lymphocytic choriomeningitis) or bacterial abscess. Attention – abscess is a contraindication to lumbar puncture, it is diagnosed exclusively by imaging methods! Rarely, it may also have an autoimmune cause and follow a viral illness or vaccination.

Multiple sclerosis

Acute vertigo of central origin lasting more than 24 hours, especially in younger people (typically in the 2nd-3rd decade) and if it occurs repeatedly can be the first symptom of multiple sclerosis. In the anamnesis, it is necessary to look for deterioration of vision, paresthesias of the limbs, in objective findings for signs of a lesion of the pyramidal tract. During the course of the disease, ataxia of cerebellar or sensory origin may appear, relapsing at first, and permanent in more advanced stages.

Migraine dizziness

Dizziness can be a manifestation of basilar migraine and so-called vestibular migraine. Basilar migraine occurs more often in young girls and is manifested by symptoms of dysfunction of the vertebrobasilar basin (vertigo, ataxia, dysarthria, perioral paresthesia, scotoma, amnesia or impaired consciousness). Vestibular migraine manifests as acute vertigo with a sudden onset typically lasting minutes to hours. Vertigo may precede, follow, or occur simultaneously with a headache. In 1/3 of cases, vertigo can occur even without a headache. Patients tend to have an increased tendency to motion sickness outside of attacks.

Cervicogenic vertigo

Cervicovestibular syndrome is a very common diagnosis, although it is a controversial entity. Disorders of the cervical spine can **cause vertigo by** several mechanisms - compression of the vertebral artery, degenerative changes, where tilting of the head is particularly risky, and proprioceptive disorders in the neck muscles and ligaments. The posterior cervical sympathetic syndrome hypothesis is now considered obslete. Cervicogenic vertigo tends to be paroxysmal, provoked by a change in the position of the head, lasting several hours. The vestibular syndrome has a central character, rather it is a feeling of instability, there is no nystagmus. **Diagnosis** is based on the finding of functional blockage of the cervical spine and the positive effect of mobilization. If we are not sure of the diagnosis, it is necessary to rule out a more serious cause of the patient's problems (carry out CT brain and USG of cerebral arteries).

Diffuse brain lesions and other causes

Central vestibular syndrome or a feeling of instability can also be a symptom of diffuse white matter lesions: demyelinating, vasculitic, post-inflammatory, but most often in **vascular-ischemic changes** of atherosclerotic etiology. This is one of the most common causes of instability in old age. Patients complain of chronic non-rotational vertigo aggravated by head movement and vertical standing (orthostasis). The condition fluctuates with the state of hydration and cardiovascular output and may progress to a picture of typical VBI.

Also, brain **tumors**, most often in the parieto-temporal location, can be manifested by recurrent attacks of vertigo lasting several minutes, sometimes accompanied by amnesia and disorientation. Rarely, vertigo can also have an **epileptic origin** in foci in this location.

Tab. 2: Differential diagnosis of vertigo

	Progress	Type of disability	Disorder	Examination

Vertigo	Isolated prolonged attack	Central	brainstem/cerebellar infarction	MRI
			cerebellar hemorrhage	CT
			multiple sclerosis	MRI, cerebrospinal fluid
		Peripheral	labyrinthine infarction	CT, ENT
			bacterial osteomastoiditis	CT, ENT
			vestibular neuronitis	ENT
	Recurrent spontaneous attacks	central (accompanying neurological symptoms)	VB insufficiency	MRI, USG
			multiple sclerosis	MRI, cerebrospinal fluid
		peripheral (accompanied by hearing impairment)	Meniere's disease	ORL
			Otosclerosis	ORL
		without accompanying symptoms	Cervicocranial syndrome	rehabilitation effect
			Migraine	trial with an antimigraine
	Repetitive positional vertigo	hearing impairment, posterior fossa symptoms	tumor of the cerebellar corner	MRI, BAEP
		a normal finding	benign positional vertigo	Dix-Hallpike manoeuvre

Cerebellar disorders with predominantly chronic development

Paraneoplastic involvement

It typically develops over months. We think of it in patients with an already diagnosed tumor, but it can also be the first symptom of an oncological disease. The most common tumors causing paraneoplastic involvement are: small cell lung cancer, breast cancer, ovarian cancer and Hodgkin's lymphoma. The examination procedure includes: the search for a primary tumor, the exclusion of metastatic disease by imaging and the exclusion of Wernicke encephalopathy(tendency to malnourishment of oncology patients). Examination of paraneoplastic antibodies can help: anti-Yo (breast, ovary), anti-Hu (small cell lung cancer), anti Ri (breast) and the finding of pleocytosis and protein elevation in the cerebrospinal fluid. Neurological impairment sometimes improves after tumor removal.

Autoimmune disorders

Patients with celiac disease, autoimmune thyroiditis, and anti-GAD (glutamic acid decarboxylase) syndrome may present with a slowly progressive cerebellar syndrome that is most likely caused by autoantibodies and in some cases responds well to treatment with intravenous immunoglobulin. Detection of the relevant antibodies (anti-gliadin, anti-transglutaminase, anti-TPO, anti-thyroglubulin, anti-GAD) is used diagnostically.

Tumors of the posterior fossa

The clinical picture depends on the rate of growth of the tumor, we usually encounter the progression of cerebellar syndrome over the course of weeks to months, and the acute condition caused by perifocal edema is no exception. Sometimes only headaches and repeated vomiting may be the only symptoms, with vermis tumors only ataxia of walking. Tumors located in the hemisphere cause hemiataxia of the limbs, sometimes vertigo with nystagmus or symptoms from brainstem compression (cranial nerve disorders or impaired consciousness during the development of hydrocephalus. **Astrocytomas** and **medulloblastomas** predominate in childhood. Metastases far predominate in adult patients, disability is less common than **hemangioblastoma**, meningioma or **ependymoma**. Diagnostics consists of imaging methods, supplemented by histological examination in indicated cases. The therapy of choice is the surgical removal of the tumor, the second option is stereotactic radiotherapy with a gamma knife. If the development of edema is suspected, corticoids must be administered.

Toxic cerebellar degeneration

It occurs most often in chronic **alcoholics**, most likely due to nutritional deficiency. The clinical picture is dominated by ataxia when standing and walking, followed by associated findings of polyneuropathy or signs of a diffuse lesion of the nervous system. Signs of acute Wernicke encephalopathy should be looked for. CT and MRI can show cerebellar atrophy. The therapy is abstinence from alcohol, adequate nutrition and long-term substitution of B vitamins. Cerebellar degeneration can also be caused by long-term administration of the antiepileptic drug **phenytoin**.

Hereditary disease

The group of autosomal-dominant spinocerebellar ataxias (SCAs) begins in adulthood with a slowly progressive cerebellar syndrome, in some cases accompanied by polyneuropathy, **extrapyramidal syndrome**, or cognitive deficit. The most common units can be proven by genetic testing, and imaging methods demonstrate **cerebellar atrophy**. Therapy is only rehabilitation, genetic counselling is important. In the differential diagnosis, it is necessary to distinguish **the cerebellar type of multisystem atrophy**, which is possible by proof of autonomic impairment and MRI findings. Sometimes, however, only the next course decides.

Autosomal recessive **Friedreich's ataxia** is the most common hereditary ataxia. In most cases, the difficulties begin in childhood, but they can also begin in adulthood. The clinical picture includes cerebellar syndrome, posterior cord syndrome, pes cavus, kyphoscoliosis of the spine, and pyramidal irritation phenomena on DK. Wilson's disease must be ruled out for any progressive cerebellar involvement with onset under the age of 45, especially if it is accompanied by extrapyramidal symptoms.

Malformation of the posterior fossa

Developmental abnormalities of the posterior fossa can cause a cerebellar disorder in adulthood. The most common is the **Arnold-Chiari malformation** (type I) with a caudal displacement of the cerebellar tonsils into the foramen magnum. In addition to cerebellar ataxia of gait, it can be manifested by obstructive hydrocephalus (headache, vomiting), compression of the trunk (vertigo, dysphagia, dysarthria) and syringomyelia (disturbance of thermal and algic sensation). It is diagnosed by imaging methods.

Tab. 3: Differential diagnosis of cerebellar syndrome

	Progress	**Disorder**	**Examination**
Cerebellar syndrome	acute	cerebellar hemorrhage	CT
		Wernicke's encephalopathy	Administration of thiamine
	the progression of the month	Paraneoplastic involvement	Brain CT, anti-Yo antibodies, neolasia screening
		posterior fossa tumors	CT, MRI
	progression years	toxic impairment	CT, usus alcohol, phenytoin
		hereditary disability	Genetics
		Arnold-Chiari malformation	CT

Symptomatic therapy

Dizziness is subjectively a very unpleasant feeling, repeated vomiting can also lead to dehydration and mineral breakdown. Symptomatic therapy is therefore indicated for acute impairment. The basic preparation is the phenothiazine neuroleptic *thiethylperazine* (Torecan), which acts as an antivertiginosis and antiemetic, it can be administered intramuscularly or intravenously, suppositories and tablets are also available. An undesirable effect, especially in elderly patients, may be the development of extrapyramidal syndrome, therefore it should not be used in patients with Parkinson's disease. Another option is to use *diazepam*, which is available in the same form as thiethylperazine. For less intense vertigo, an antihistamine such as *embramin* (Medrin), a combined preparation, can be used *Arlevert* (dimenhydrinate + cinnarizine), which is only available in tablet form, or *promethazine* (Prothazine), which is also available in injectable form. Adverse effects are depression, drowsiness and anticholinergic effect (blurred vision, tachycardia, dry mouth, constipation, micturition disorder).

For chronic vertigo caused by ischemia of the labyrinth or Meniere's syndrome, the drug of the first choice is the antihistamine *betahistine* (Betaserc), which is used in a sufficient dose (initially 48 mg per day) for a sufficiently long time (at least several weeks). *Cinnarizine*, which is also effective in preventing motion sickness, can have a certain effect but can worsen Parkinson's syndrome in older patients, and *ginkgo biloba* extract can increase the risk of hemorrhage in anticoagulated patients.

Antivertiginosis should not be used in the recovery phase from an acute vestibular syndrome, as they delay compensation. Furthermore, it should not be used in chronic bilateral vestibular lesions, when the most important therapy is vestibular rehabilitation with the training of compensatory strategies using the somatosensory and visual apparatus.